patient had the movements of St. Vitus' dance, and at other times showed athetosis in both arms and legs, and at still others merely the twitching of epilepsia continua. The explanation of this variability in type of motor disorder probably is that the character of the movements depends largely on the intensity of the irritation as well as on the area covered by the lesion.

The important thing in diagnosing the condition is to find the underlying cause. The diagnosis of brain tumor in my case was based entirely on the occurrence of the symptoms of a focal brain lesion in a person suffering from carcinoma. It is always safe to diagnose cerebral metastasis in any case of carcinoma in which cerebral symptoms occur if hemorrhage and thromosis can be excluded. The absence of choked disk, cerebral vomiting, vertigo, and headache do not exclude tumor, carcinoma, or any other malignant growth being present elsewhere in the body, and the presence of focal symptoms points strongly toward it because the classical symptoms of tumor occur only when the growth has attained some size, but a very small tumor may cause focal symptoms if situated in any part of the cortex having specialized functions.

PULSUS ALTERNANS.

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In 1872 Traube, in an admirable article, directed attention to a hitherto undescribed condition of the pulse, with rhythmical irregularity in volume, which he called pulsus alternans, the sphygmogram showing waves of large and small amplitude, following each other in regular succession. He also noticed in the case which he described that while large and small beats were placed alternately throughout, the interval which separated the large from the small beats exceeded that which separated the small from the large beats. He further observed that the rhythm of the heart's action was perfectly normal.

Despite this early publication of Traube no other case of a similar character was recorded until 1892, when Sansom, in his book on the *Heart and Aorta*, refers to a case of cerebral hemorrhage, Cheyne-Stokes respiration, and general arteriosclerosis, with radiograms showing typical pulsus alternans. He states that the peculiar derangement of the rhythm of the pulse as seen in the sphygmogram was not at any time detectable by the finger. It is an example of alternating pulse, a more ample one being always succeeded by a less ample tracing, each beat being perfect in all its elements, the irregularity being in volume only, and the cyclic sequence being invariable. Sansom says, to explain the large followed by the small pulse, we must assume that the ventricle delivers a full content in the large beat and an imperfect content in the small beat. The nervous mechanism which regulates this occurrence is the vagus. The lesion affecting its origin in the floor of the fourth ventricle is one which, calling into play its function of inhibiting the heart's movements in slight degree, is insufficient to elongate the diastolic pause, but antagonizes the sympathetic incompletely, so that this influence of the latter comes into play too soon in every alternate cycle. He also refers to another case of clinical alternation occurring with the onset of Cheyne-Stokes breathing. Hering believes that both the experimental and clinical alternation is to be explained on the hypothesis of a hyposystole or partial asystole. He has observed in the exposed hearts of lower animals that under pathological conditions all the heart muscle fibers are not in the same degree of contraction; that during the smaller beat of alternans, different degrees of contraction of the ventricle occurred. In this study Hering also showed that when tracings were taken from the clinical apex, or apex of the left ventricle in animals, the larger wave of the cardiogram corresponded to the smaller wave of the radiogram, thus producing a distinct incongruity between the apex beat and the pulse curve. On the other hand, when the cardiogram was taken from the third left intercostal space, or over the body of the right ventricle, the highest upstroke of the cardiogram corresponded to the strong beat of the radial. These results were constant and repeatedly observed. After the death of the animals needles were inserted through the intercostal spaces at the points where the cardiograms were taken, and on opening the thorax it was found that the needle in the fifth intercostal space was lodged in the left ventricle just above the apex. The needle in the third intercostal space was found in the conus of the right ventricle.

Hering believes that this incongruity between the cardiograms from the apex and those from the conus region, and the radiograph curves in alternans shows that the musculature which causes the apex beat has little or no propelling effect on the contents of the left ventricle. He further states that alternation is due to a disturbance of contractility without disturbance of rhythm, rapidity of rate, or lack of stimulus production, but is dependent on pathological changes in certain areas of the myocardium, the smaller beat being an evidence of the fact that the diseased areas of the myocardium react to stimulus production but slightly or not at all.

These observations of Hering were verified and somewhat elaborated by Rihl, who in 1906 reported 4 cases of clinical alternation, with two autopsies. All 4 cases were accompanied by general arteriosclerosis, chronic myocarditis, and hypertension. In the

3 cases that came to autopsy marked myocardial changes were found associated with general arteriosclerosis and chronic interstitial nephitis. In one case the coronary arteries were greatly sclerosed and the seat of aneurysmal dilatation. Rihl found the cardiac rhythm perfectly normal. He found that the height of the apex differed, a large and small beat following each other in regular succession. This shows alternation in the left ventricle. None of his cases showed evidence of alternation of the auricles. In all his cases an occasional extrasystole was observed, after which in each instance the alternation was more pronounced. He also found that anything that caused the heart to beat more rapidly, such as walking or other exercise would make the alternations more marked.

In 1905 Volhard discussed clinical alternation, and published 2 cases of true pulsus alternans and 2 cases of pseudopulsus alternans or continuous bigemina. The cases of true alternans were in young men, one a case of mitral stenosis with decompensation, aged twenty-seven years, and the other a case of chronic Bright's disease with albuminuric retinitis, aged twenty-five years. Both died within a few weeks after entering the hospital. His conclusions were that in man cardiac alternation is due to variation in strength of the contraction of the auricles as well as the ventricles; that the change in the pulse is due to difference in volume and that the cardiac rhythm is absolutely normal; that the smaller wave of alternans is due to the weakened contraction of the small systole plus the high aortic pressure; that with extrasystoles the alternation is very much pronounced; that continuous bigemina, or pseudopulsus alternans, resembles closely true alternans, but may be distinguished by the long compensatory pause following the small wave, and the small wave occurring at its normal time period.

Engleman has shown from experiments on lower animals that alternation is due to disturbances of contractility and not conductivity. Wenckebach is of the same belief and divides pulsus alternans into two forms: In one the time of the small wave exceeds that of the large, and the other in which the time interval of the small wave is the shorter. The first form he explains by the fact that the small wave is transmitted more rapidly into the aorta than is the larger; and the second form, with the large time interval of the small wave, by the hypothesis that alternation is due to weakened or exhausted contractility.

Hoffman believes that, owing to the disturbed function of contractility existing in alternation, contraction occurs more quickly than normal, and hence the periods of time of the systole and diastole are necessarily both shortened. In other words the heart muscle reaches the point of maximum contraction and relaxation much more quickly than in health.

Mackenzie, in his book on *Diseases of the Heart*, second edition, states that a striking and characteristic sign of exhausted contractility is sometimes seen in the size of the pulse beat. He further states that "I have repeatedly dwelt upon the fact that the deviation and force of contraction depend in a measure on the length of the preceding period of rest. In hearts with good contractility, recovery after a contraction is so rapid that little or no difference can be detected in the size of the beat after pauses of varying duration. In cases of exhausted contractility, on the other hand, recovery is slow, so that the size of the beat has a distinct relation to the length of the preceding diastolic pause. This is exemplified in cases of extrasystoles. In those with good contractility the succeeding beat following the compensating pause may not be much increased, or may be very large, and the succeeding beats may be almost uniform in size. Whereas the same phenomenon occurring in hearts with exhausted contractility, the large beat following the diastolic pause, may be followed by several smaller beats of varying size in regular alternation. This difference in the size of the beats is an important one, as it indicates a grave exhaustion of contractility. Not infrequently the difference in the size of the beats persists for a shorter or longer period in such manner that the large beat alternates with the small beat, the pulsus alternans.

Mackenzie explains the pulsus alternans as follows: When contractility is depressed, if time be allowed for a full and strong contraction, the longer duration of contraction encroaches upon the period of rest, so that by the time the next stimulus arrives the contractility has not sufficiently recovered and a smaller and shorter contraction occurs. As this contraction is shorter in duration the period of rest is thereby lengthened before the next stimulus arrives, so that the contraction would be stronger and larger; being larger it will again encroach upon the period of rest and the process of alternation goes on.

Of the more recent studies on this complex cardiac mechanism those of Davenport, Windle and Thomas Lewis are instructive and interesting. Windle states that pulsus alternans is properly given only to that form of pulse in which a strong beat is regularly followed by a weaker one, the duration of the pulse periods being generally equal, although at times there may be slight alternation in the rhythm of the beats as well as in the force; but the difference in duration from one pulse to the next is small, and invariably the larger period belonging to the stronger beat, the weaker beat being therefore slightly delayed. From the polygraphic curves taken from his twelve cases, Windle states that he observed but one example of alternation of the clinical apex, while in all disparity in force of alternate radial pulses was decided and at times extreme. In the exceptional case the stronger beat of the heart curve coincided with the weaker one of the pulse. Thus this example confirms

Hering's observations. He also offers in explanation of this apparently incongruous phenomenon, Hering's theory that the musculature which forms the apex is not concerned in propelling the ventricular contents into the aorta. In this latter case alternation was often pronounced in records of the jugular pulsation; invariably the large *a* wave corresponded to the higher of the radial pulse beat. He observed no variation in intensity of the pitch or duration of the heart sounds or murmurs coincident with alternation of the pulse. His observations lend support to the view of Hering that alternation of the heart beat may be due to a partial systole of the ventricle. Lewis states that heart alternation occurs under two circumstances: (1) when the cardiac muscle is not altered structurally, as an accompaniment of great rapidity of rate; (2) when the pulse is of normal rate and under such circumstances the muscle is markedly degenerated.

Lewis' experiments were upon rabbits with ligature of the coronary arteries. He found that obstruction of one or other coronary artery was followed by rapid and new rhythms arising in the ventricle, the ventricular rhythm dominating that of the cardiac chambers, and that if the heart rate is greatly increased, alternation frequently appears both of the ventricles and auricles independently. As a result of these experiments, Lewis observed a divergence between the alternation in the ventricle and carotid, and explains this phenomenon on the coexistence of alternation in the force of the auricular contractions, thus supporting the view of Volhard, whose tracings showed that the large apical curve corresponded to the smaller radial beat alternation occurring in the amplitude of the *a* waves in the jugular, the small radial beat being preceded by the small *a* wave.

Lewis further states that the electrocardiographic curves obtained in clinical alternation are similar to those obtained experimentally, there being a divergence between the heights of *R* and *T* waves and the amplitude of the radial upstrokes. These facts demonstrate the identity of clinical and experimental alternation.

From a careful survey of the above-mentioned observations one is led to the opinion that both clinical and experimental alternations is the result of varying degrees of impairment from exhaustion of that most important function of the myocardium, contractility, which may be due to temporary exhaustion from the overwork of great rapidity of action or to permanent exhaustion and early cardiac failure, the result of grave pathological changes in the myocardium. The sequence of events of contraction of the cardiac chamber in alternation is normal. There is no defect of conductivity and the myocardium responds to stimulus production. The only abnormal rhythm is the variation in volume of the pulse beats and ventricular output, each large beat being succeeded by a smaller one, and so on indefinitely. This variation in amplitude of the pulse beat, which is an evidence of exhausted contractility is of itself, owing to the lack of time interval between the small and large beat and the greater interval between the large and small beat, a menace to the further restoration of this function, because of the marked shortening of the preceding diastolic pause, which is so essential to a full restoration of the function of contractility. It is this shortening of the diastolic pause in all cases of long-continued tachycardia that results in early cardiac exhaustion.

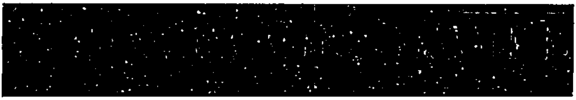

FIG. 1.—Pulsus alternans, with an occasional extrasystole. S. B. P., 230; D. B. P., 170. Chronic interstitial nephritis, chronic myocarditis, and general arteriosclerosis. Note the time interval of the small wave is the same as that of the large wave, $\frac{1}{5}$".

FIG. 2.—Paroxysmal tachycardia which continued six days, showing the alternation of acute myocardial exhaustion.

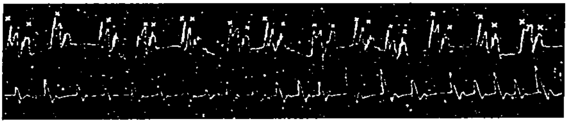

FIG. 3.—Pseudopulsus alternans, or continuous bigeminy due to extrasystoles. Mitral stenosis and regurgitation.

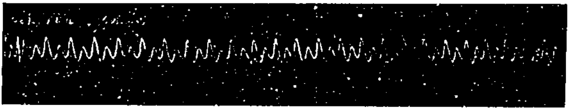

FIG. 4.—Dicrotism resembling clinical alternation, second week of typhoid.

Temporary exhaustion of contractility often occurs with the production of a typical pulsus alternans from an overtaxation of the cardiac musculature, the result of a too rapid rate, such as may occur in paroxysmal tachycardia, auricular flutter, auricular fibrillation, or the long-continued tachycardia which accompanies Graves' disease. Experimentally it may be produced in healthy hearts by electrical stimulation, the injection into the bloodstream of digitalis, antiarin, aconitin, glyoxylsäure, and hemolytic serum. It is most frequently encountered, however, when the heart is beating within its normal rate, and when it denotes a grave pathological condition of the myocardium, with failing contractility. Most of the observed cases of this type of pulsus alternans have occurred in patients with general arteriosclerosis and hypertension, associated with chronic myocarditis and chronic nephritis. It frequently comes on during a period of Cheyne-Stokes respiration and continues to the fatal issue. It is usually not observed, however, until compensation is failing and the heart chambers are suffering from dilatation, the heart fibers having undergone both fibroid and fatty changes. It is not at all uncommon in the greatly dilated hearts of mitral stenosis or insufficiency where decompensation has occurred. It also occurs during and directly after recurring attacks of angina pectoris. Mackenzie has reported a notable example of this sort, and one of the five cases of clinical alternation which have recently been under my observation occurred after a repetition of attacks of angina pectoris, and lasted but a week, when the myocardium gradually failed and the patient succumbed. In this case the systolic blood-pressure was always under 140 mm. Hg. Alternation frequently comes on during a period of Cheyne-Stokes respiration and then continues to the fatal issue.

The relation of extrasystoles to alternation is most interesting and of great practical importance; in fact, many cases of regular bigemina have been confused or mistaken for and described as true cases of pulsus alternans. This so-called pseudopulsus alternans, or continuous bigeminia, may be distinguished from true alternans by the long-time interval, diastolic or long compensatory pause following each small or extrasystolic beat, and this in turn being followed by a larger beat, while the small beat has its normal time interval; whereas in true pulsus alternans the longer period follows the large beat or the large and small beat are equidistant from each other.

In a large number of my tracings showing extrasystoles, alternation has frequently succeeded for a single or a number of beats, the large wave following the long compensatory pause after the extrasystolic contractions, thus showing a decided impairment of the function of contractility. In many of the recorded cases of true alternation an occasional extrasystole is interpolated, and following such extra beats the alternation immediately becomes more in evidence. Not only may alternation follow extrasystoles, but, as shown by Windle, it often precedes the extrasystolic contractions. Several such examples have come under my observation. While I do not ordinarily regard the appearance of extrasystoles as of evil prognostic import, I do so regard those extrasystoles which by repeated observation are found to be invariably followed by alternation extending over a considerable period of time. In two such examples of my own, death has occurred from decompensation and failing myocardium within two years.

Of the 5 cases of clinical alternation which have been under my care for the past two years, 3 have died—one at the age of seventy-eight years, of failing heart, following a series of attacks of angina pectoris, resembling a true anginoid status, with the clinical evidences of general arteriosclerosis and chronic nephritis, without hypertension. One died suddenly, when aged sixty-eight years, of general arteriosclerosis, high blood-pressure, chronic nephritis, and chronic myocarditis. A third, a man, aged forty years, died with all the evidence of decompensation, Cheyne-Stokes breathing, and uremia, the blood-pressure remaining high until near the end. The alternation in this case was first observed at the onset of the altered respiratory rhythm. The 2 cases now living are clinically typical examples of interstitial nephritis, general arteriosclerosis, hypertension, and chronic myocarditis. The older patient is aged seventy-two years and the other fifty-two years.

In 4 of these cases it was impossible to detect by palpation of the radial pulse the slightest evidence of alternation nor could one perceive by palpation of the clinical apex the slightest evidence of such a condition. In one case, however, the variation in volume between the large and small beat was so great that palpation of the radial was highly suggestive, although not positive, of alternation. The small beat resembled so closely the secondary wave in dicrotism that I was unable to distinguish between it and alternation until radiograms were taken. I therefore believe that palpation alone of any artery, radial or carotid, is a poor index of the presence of alternation or of the condition of the true action of the heart or of the height of the blood-pressure. I do believe, however, that defects of contractility from myocardial degeneration in the permanent irregularity of auricular fibrillation, or continuous bigemina, may be detected by comparing the rate of the radial pulse with that of the apex beat, determining the latter either by palpation or ausculation, and noting the deficit between the rate of the apex and that of radial. It is no uncommon observation to find that a third or half of the systoles of the left ventricle fail to be registered at the wrist. Many of the systolic contractions are so feeble that the intraventricular pressure does not overcome that of the aorta, and therefore the aortic valve is not lifted. If, however, the aortic pressure is overcome the pulse fails to reach the wrist because of the high blood-pressure plus the peripheral resistance.

In my cases of alternation no change in the intensity, pitch, or quality of the heart sounds were detectable. An extra or interpolated third sound was never heard. In one case, with a relatively loud systolic apex murmur, no change in its timbre occurred. In 3 cases no marked alternation was observed in cardiograms taken from the clinical apex. In 2 cases, however, such alternation was very distinct. The respiratory excursions, because of the dyspnea in individuals with myocardial insufficiency, is so great that it is often difficult without temporary cessation of the respiratory act to get satisfactory cardiograms. In all cases the rhythm of the heart's action was normal. In three of the cases where satisfactory phlebograms were obtained the *a-c* interval was not increased, showing that conductivity was normal.

From this study the following conclusions may be drawn:

1. That pulsus alternans, while not common, is not very rare. I agree with Mackenzie and Windle in giving it the third place among pulse irregularities, extrasystoles being placed first and the persistent irregularity of auricular fibrillation the second.
2. Clinical alternation is frequently overlooked because it cannot be recognized without the graphic registration of the pulse.
3. Its presence is positive evidence of defects of the function of contractility, this in turn being due to temporary or permanent myocardial changes.
4. From a prognostic stand-point the presence of pulsus alternans, save the temporary form, due to great rapidity of rate, is grave, as it indicates extensive myocardial degeneration, most patients dying from cardiac insufficiency within three years of its onset.
5. Extrasystoles, invariably followed by alternation, also indicate exhaustion of contractility and carry with them a grave prognosis.
6. Persistent false bradycardia, due to feeble systoles, where the deficit between the apex beat and the pulse rate is but little altered by the exhibition of cardiac tonics, such as digitalis or strophanthus, also indicate grave defects of contractility, with extensive myocardial degeneration and unfavorable prognosis.

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